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LANDRY'S PARALYSIS: REMARKS ON CLASSIFICATION.

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In spite of the fact that the complex of symptoms known as Landry's paralysis has been, during recent years, the subject of much critical work, there appears still to be opportunity for an analysis of the claims of the affection to be recognized as an independent disease. We offer the following case, therefore, typical in its clinical course, rather as a text for what is to follow than as a contribution of value to the extensive literature of the general subject:

The patient was a man of fairly strong physique, fifty-two years old. He was admitted to the Long Island Hospital, Boston Harbor, October 7, 1895. The antecedent history is unimportant as bearing on the present condition; he was not addicted to alcohol, and had had no venereal disease.

One month before entrance to the hospital the patient had had what he supposed was malaria, from which he recovered. Five days before entrance he was as well as usual. At about that time he stumbled and fell, without loss of consciousness. He was taken home, feeling weak. That night he had considerable pain in the back, and dorsal leg muscles. There was no headache. The chief complaint was a sense of weakness with occasional extreme pain, particularly in the legs. There was no bladder disturbance. A few days later he fell again in attempting to get off a car. He had, at this time, the same feeling of weakness he had before experienced, but was able to walk about one hundred yards with the aid of a cane. The sense of weakness increased, and he was finally admitted to the hospital. On entrance he was found to be suffering from general weakness, considerable pain and a sensation of cold. The cranial nerves were at this time uninvolved. He remained essentially the same until October 9. At noon of that day a difficulty in articulation came on, then a gradually increasing disability to close the eyes; the following night he had some regurgitation of food and considerable frontal headache. Taste, smell and sight were unaffected; there was no diplopia. The general weakness was now so great that he was unable to stand unsupported. He complained of a numb sensation about the region of the stomach, and said that "his skin felt dead," below the level of the umbilicus. Bowels open.

Physical Examination: October 10. *Motion:* Excessive weakness; active and passive movements of the extremities free, but active movements executed with difficulty. Knee-jerks not obtainable; plantar reflexes absent; cremaster absent on the right and present on the left; abdominal and epigastric lacking. Arm reflexes normal. Considerable fibrillary tremor in the adductors of both sides. The patient is emaciated.

Sensation: Subjective complaint of cold, especially about the ankles. No pain on deep pressure over the nerve trunks; slight tenderness over the tendons of the dorsal thigh muscles. Sensation in the legs possibly slightly diminished to pin prick, but the point and head are correctly distinguished.

Cranial Nerves: Hearing, sight and smell unimpaired. Movements of the eyes free in all directions; the pupils react normally to light. Double facial paresis, more marked on the right side. Tongue protruded straight, but cannot be projected beyond the lips; swallowing imperfect; speech markedly affected, but no true aphasia. No mental disturbance whatever.

Heart: No murmurs; rapid; pulse 120.

Urine: Negative. *Temperature* normal.

Five days after entrance (October 12) a second examination was made in consultation with Dr. James J. Putnam. The general weakness had increased and slight sensory disturbances in the legs were noted, characterized by delay in perception and a failure to distinguish between hot and cold. The hands and face showed no abnormality. There was slight ataxia of the arms, but the hand-grasp was firm. The spleen was apparently not enlarged. (See autopsy note.) Electrical examination essentially negative.

October 17. Pulse 120-132; weak; tracheal râles. Speech more affected than at the last visit, but in the same manner.

Double facial paresis practically complete. Eyes glassy; cold sweating; urinary and fecal incontinence. Sensation to pin prick apparently still preserved; motor weakness extreme. Mental state clear; he is hopeful as to his condition. Died October 17, the same day, ten days after entrance to the hospital, and fifteen days after the first onset of symptoms.

Autopsy: 36 hours after death, October 19, under somewhat disadvantageous conditions. The body was considerably emaciated. The cord, brain, viscera, and following peripheral nerves were examined: the left pneumogastric, a portion of one sciatic, the left popliteal.

The cord microscopically showed no abnormal appearances; the membranes were normal. The skull and brain were normal, except for considerable opacity over the convexities, to which no pathological significance is to be attached. There was no increase in the amount of cerebro-spinal fluid.

Cultures were made at autopsy from the popliteal nerves and from the fluid about the cauda equina. Subsequent study of these cultures and the attempt to demonstrate bacteria in the tissues were rendered futile by the presence of contaminating organisms, probably putrefactive.

The chest cavity showed no pleuritic adhesions; the lungs were normal, except for a small focus, probably tuberculosis, at the right apex. Liver, heart and kidneys were normal in gross appearance. The spleen was considerably enlarged, weighing 360 gms. Intestines and stomach normal. No general arteriosclerosis.

Portions of peripheral nerves, a bit of the gastrocnemius muscle, the brain and cord, and a specimen from the kidney, were placed in hardening solutions preparatory to microscopic examination.

Microscopical Examination:

Peripheral Nerves: Marchi preparations: The sections examined show no characteristic staining of the myeline. The contour of the nerve fibers may often be made out, but the varicosities and stained fat droplets as seen in pathological conditions are absent. The same nerves stained by the Weigert method show considerable irregularity of staining. In view of the fact, however, that Marchi sections are negative and that the outlines of the Weigert stained fibers are regular, together with the relatively long time after death at which the specimens were hardened, lead us to think that what abnormal appearances are present are due to artifact, and are in no way to be regarded as pathological. Ventral nerve roots near the periphery of the cord show no abnormality. Dorsal nerve roots normal.

Spinal Cord: Sections cut from a large number of levels and stained by the usual methods. Unfortunately for the completeness of this report the Nissl method was not used, owing to the fact that the period after death at which it was possible to make the autopsy we felt too great to permit of definite conclusions being drawn from changes in the constituents of the cell body. The white matter of the cord as a whole shows no change whatever, so far as the nerve elements are concerned. The gray matter appears normal, except for a considerable degree of hyperpigmentation of the cells of the ventral horns, best brought out by staining with acid-fuchsin in aqueous solution. Many of the cells are almost completely filled with pigment, leading to an entire obliteration or marked excentric position of the nucleus and to a loss of cell structure. For example, in one group of twenty-five cells in the cervical region, ten show no trace of normal nuclei. Other cells of the same group are entirely normal in appearance, with well preserved processes and no dislocation of nuclei toward the periphery. The contour of these cells is regular, and many of them are noticeable from the fact of the small amount of contained pigment, in contrast with their engorged neighbors. So far as one may judge from this method of staining, the nuclei and chromatic portions of these cells are normal. This is, however, a point upon which we evidently cannot be dogmatic.

Blood Vessels: There is no evidence of arteriosclerosis; neither the larger nor smaller vessels of the cord show any true thickening of their walls. The smaller arteries throughout the cord, both in gray and white matter, are in many cases surrounded by hyaline material staining purple with hematoxylin, and remaining unstained with fuchsin and Weigert hematoxylin. This hyaline material apparently does not represent a degeneration of the vessel walls, but rather an extraneous deposit. In some instances the appearances are strongly suggestive of thrombi plugging the small arteries; in others the hyaline mass is evidently outside the lumen. There is also a certain amount of this same material lying free in the tissue and about the periphery of the cord; such deposits may occasionally be seen in the immediate neighborhood of nerve cells, in other respects apparently normal. There is no evidence of inflammatory change either in gray or white matter. The pia is normal in all the sections studied, excepting in the mid-thoracic region. At this point there is a typical tuberculous meningitis, involving the dura very slightly, but the pia throughout its whole circumference. The pia is everywhere thickened and infiltrated, the blood vessels are involved, and the nerve roots, both ventral and dorsal, are invaded to a greater or less degree by the new-formed tissue. Giant cells and typical areas of necrosis are apparent. The periphery of the cord is invaded to a slight degree in typical fashion, but not sufficiently to interfere with general cord functions. We are unable to say how far downward this focus of tuberculosis extended, owing to the imperfection of the serial. There is no evidence of tuberculosis in the cervical region, nor in the oblongata, pons, cerebellum or cortex cerebri, so far as we have been able to study them, from the specimens at present at our disposal.

Oblongata and Pons: Nothing abnormal found.

Cerebellum: No pathological change discoverable, beyond the appearance of very numerous, usually round bodies, without concentric markings, and staining similarly with hematoxylin to those described in the cord, but much more regular in outline. These bodies are located in greatest numbers in the immediate neighborhood of blood vessels.

Cortex Cerebri: Similar structures are numerous, resembling those described in the cerebellum more closely than the irregular masses found in the cord. The cortex itself presents nothing abnormal.

Spleen: Simple hyperemia.

Kidney: Considerable degree of granular degeneration.

The search for bacteria as possible cause was unavailing, owing to contamination.

A summary of the findings in this case gives as positive result the presence of numerous hyaline bodies (amyloid) in the central nervous system, a focus of tuberculous meningitis in the thoracic cord, and changes of a pigmentary character in numerous ventral horn cells, studied particularly in the cervical region. The investigation is negative, as regards the discovery of degeneration in peripheral nerves, or positive evidence of the presence of bacteria as an exciting cause. Further changes in nerve cells than those noted above may have existed, but lack confirmation, because the Nissl method was unavailable.

An analysis of the lesions found, in the light of our present knowledge of so-called Landry's paralysis, will be of interest.

Hyaline Bodies: In 1891, E. Klebs[1] described a case of

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[1] E. Klebs. "Ueber Landry'sche Paralyse." Deutsche med. Wchschft. Vol. 17, 1891, p. 81.

Landry's paralysis, in which the essential lesion found was a hyaline thrombosis involving only the branches of the ventral spinal artery to the ventral horns. The nerve cells he found surrounded by a net-like substance, containing cellular elements. This he regarded as a transudation following thrombosis. The situation of the thrombosis only in the region of the ventral horns, he would explain by the anatomical arrangement of the vessels of the cord. The patient had a tuberculous pericarditis, resulting from perforation into the pericardial sac, of a caseous bronchial gland. The etiology of the thrombi is left in doubt; no tubercle bacilli were found in the vessels.

This case, which, so far as we know, is the only one of the sort in the now extensive literature of Landry's paralysis, is of interest in connection with the markedly hyaline condition of the smaller vessels observed in our case. As stated in the description of the microscopic appearances, the hyaline was in our case not so sharply limited to the distribution of a single artery as in Klebs' cord, since very similar conditions were found also in the cerebellum. Although in many instances there is in our specimens a strong suggestion of thrombosis, it is evidently not so conspicuous as in Klebs', judging from his description. The presence of hyaline-like material in the region about vessels, and also about nerve cells, is possibly what Klebs speaks of as a transudation following thrombosis. In any case the hyaline deposit is of the nature of an infiltration rather than of a degeneration of vessel walls. The larger vessels are not involved. Of interest also is the fact that in both Klebs' case and ours there was a latent tuberculosis, certainly in our case not sufficient to cause death, or symptoms of moment, though possibly associated in some way as yet unknown with the extensive hyaline formation. Admitting, as Klebs seems inclined to do, that there is a definite connection between the hyaline and the symptoms of ascending paralysis, we are at a loss to see how, in our case, such a relationship is to be maintained, inasmuch as the hyaline was not sharply limited to the ventral horns, and in any event cannot be conceived as productive of the rapidly fatal symptoms observed. A more reasonable supposition is to regard the hyaline changes as part of a general process of as yet unknown etiology, and therefore an effect rather than a cause. It should be mentioned that at least two other writers, Baumgarten and Immermann, allude to the existence of hyaline deposits in cases of Landry's paralysis.[2] A paper by Spiller[3] is of interest also in this connection. He found a great number of amyloid bodies in a man of forty, dying of a disease resembling Landry's paralysis, and suggests that possibly we have not attached sufficient significance to the part such bodies may play in pathological processes. Certain of the bodies found in our case are not unlike those pictured by Spiller in his figures 3 and 4.

Tuberculous Meningitis: We have described a localized tuberculous meningitis of the cord in the thoracic region, with no other discoverable lesion of the same character in the specimens now at our disposal. A lesion of the lung, presumably of tuberculous origin, was also mentioned. The association of a tuberculous meningitis with the symptoms of ascending paralysis has not before been described, and very probably is to be looked upon merely as a coincidence. A review of previously reported cases shows the occurrence of tuberculosis in several instances, but certainly not in a sufficient number to be regarded as standing in constant causative relation to the symptoms. Nevertheless, in the light of the negative researches regarding etiology, it is not to be forthwith denied that the infection with tuberculosis may not in a predisposed individual lead to the outbreak of Landry's paralysis, in the same sense that syphilis does to tabes dorsalis. We have, however, no positive evidence, nor analogy, to substantiate this view, and we could, at best, merely regard tuberculosis as one factor in a multiple etiology.

Nerve Cells: We have noted changes in numerous cells of the ventral horns, which are not to be explained as senile (the patient's age was fifty-two), nor as due to the long period which elapsed between death and the fixation of the material.

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[2] J. J. Thomas. "Two Cases of Acute Ascending Paralysis with Autopsy." Am. Jour. Med. Sciences, Aug. 1898, p. 133. This paper contains an excellent digest of the literature up to 1898.

[3] Spiller. "On Amyloid, Colloid, Hyaloid and Granular Bodies in the Central Nervous System." N. Y. Med. Jour., Aug. 13, 1898.

The changes are therefore positive, and may stand in causal relation to the symptoms, though we are more and more of the opinion that a conservative attitude toward the significance of cellular changes, such as are shown, for example, by the Nissl method, is in the present state of our knowledge the safest one to adopt. Pigmentation is notoriously a dangerous index of cell changes, since pigment is a perfectly normal constituent of many cells in the central nervous system. We are, however, safe in attributing to it a pathological significance, when it so predominates as to completely fill the cell body, particularly if associated with this hyper-pigmentation there be also a marked dislocation of the nucleus. The further and more important question of how such pigmentary atrophy is associated with the peculiarly rapid and characteristic symptoms of ascending paralysis remains absolutely unanswered, and we have no desire whatever to force the relationship. The point to be noted merely is that such cell changes existed in a nervous system which was also the subject of ascending paralysis.

The peripheral nerves, as far as our examination went, remained intact. We must, therefore, conclude that the lesions found in our case, however interesting from a general point of view, afford us little help in the interpretation of the symptom-complex observed during life, a result to which many other examinations of this so-called disease have also led. It is clearly one thing to find lesions, and quite another to associate them causally with symptoms. This is an error into which we are likely to fall, as our enthusiasm for the mere findings of the microscope increases.

It is quite superfluous to rehearse again the literature of Landry's paralysis. That has recently been done with close critical analysis by Diller and Meyer,[4] Bailey and Ewing,[5] Mills and Spiller,[6] and Thomas.[7] No doubt much has been gained by this careful work, and by other papers which have since been published, but still the opinions differ fundamentally as to the exact or even approximate nature of the disease, or whether, in fact, it is after all to be regarded as an independent disease process or not. When such a diversity of opinion prevails at the end of a long period of conscientious study, we may assume that the various writers are using terms inconsistently, and with different meanings, or that we have not yet arrived at a fundamental conception of the process which lies at the root of the clinical symptoms, and hence are giving superficial facts an importance which does not belong to them. In the study of Landry's paralysis we have apparently fallen into both these errors.

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[4] Diller and Meyer, Amer. Jour. Med. Sc., April, 1896.

[5] Bailey and Ewing, New York Med. Jour., July 4, 11, 1896.

[6] Mills and Spiller, JOUR. OF NERV. AND MENT. DIS., June, 1898.

[7] Thomas, Am. Jour. Med. Sc., Aug., 1898.

Landry in 1859 described a series of symptoms, chiefly motor in type and usually fatal, which were quickly received into the long list of diseases as an individual entity, without pathological or etiological basis. The inevitable occurred. Conditions which showed pathological changes, with clinical course similar to that described by Landry, were still called Landry's paralysis, doubt merely being thrown on the earlier negative findings. These cases, which varied considerably from Landry's original type, were included in the same category, and the whole discussion about peripheral neuritis arose. If we mean by Landry's paralysis what Landry described, it is evidently absurd to arbitrarily extend the meaning of the term on the clinical side to various conditions which he did not describe. If the word is to be used at all as descriptive of a supposed disease entity, it must be used in the sense Landry meant it to be used and in no other, otherwise it would be far more conducive to progress if it be immediately dropped. Even a superficial study of the literature shows that the various writers on the subject have been absolutely inconsistent in the use of the term, "Landry's paralysis." The tendency has been to make the symptoms fit the pathological findings as these have grown more conspicuous and numerous, with what complete confusion we know. In other words, if Landry described a certain disease, it is clear that we must find the pathological anatomy and etiology of that supposed disease, and not of some other, or else give up the word as an unnecessary encumbrance to our already over-burdened nomenclature.

The classification of the group of symptoms known as Landry's paralysis becomes, therefore, of paramount importance, if our studies are to lead to any profitable end. If we find that the various symptoms comprising the group are sufficiently coherent to make highly probable the final unity of the entire process, we are justified in maintaining the name: otherwise not. The first question to put to ourselves is: Are any of the individual symptoms described by Landry fundamental? Motor paralysis of a flaccid type beginning in the lower extremities and without involvement of the sphincters must now be looked upon as in great measure a fortuitous circumstance. Cases in which the upper extremities have been first involved have been described with, in other respects a typical symptom-complex of so-called Landry's paralysis. Leyden has seen fit to distinguish two forms, which he calls bulbar and neuritic, to overcome this difficulty of the direction of spread of the process. Hence, according to this point of view, only some cases of Landry's ascending paralysis are ascending. Landry was then wrong in laying stress upon this ascending characteristic of his disease. Certainly the flaccidity of the muscles and the non-involvement of the sphincters are of no consequence in establishing the identity of a new disease process, since they occur frequently in a variety of involvements of the peripheral motor apparatus. To go a step further, it seems that Landry was very tentative regarding sensation. In a case he described, sensation was definitely affected, and we have rather assumed that the opposite was the fact, because in many cases it has not been an obtrusive symptom. The frequently reported unaltered condition of the muscles, as regards atrophy, and the maintenance of unchanged electrical reactions are no longer to be regarded as significant of anything peculiar in the disease process beyond the fact that it is often very acute. In more prolonged cases, changes have often enough been found to throw doubt upon their absolute non-existence as a future possibility even in those cases which quickly result in death. Finally, to say that a disease is usually fatal does absolutely nothing to clear up the difficulty. It is not always fatal, we are informed, and even if it were, that fact alone would not mark it as in any way peculiar.

Evidently, then, none of the supposed classical symptoms of Landry's paralysis have, in the light of our present knowledge, anything characteristic about them. As a group they are equally vague, and have no fundamental unity as an individual process. As long ago as 1876 Westphal, apparently seeing how diverse the speculations regarding the symptom-complex were becoming, attempted to define exactly what should be regarded as Landry's paralysis. To that end he made the statement that Landry's paralysis was characterized by its progressive, ascending, and finally fatal course; by the lack of electrical changes in the paralyzed muscles, and by the negative findings at autopsy, statements which succeeding investigators at once began to modify as too narrow to include all the cases. With this reaction, Landry's paralysis, as a clinical entity, to our minds, ceased to exist. We have since been talking about various more or less coherent symptoms vaguely resembling Westphal's conception, but always modified to suit individual needs. The most interesting, and in some respects the most significant, reaction against the early ideas, has been the insistence that everything is explainable on the basis of a peripheral neuritis. Ross inaugurated this movement in 1889, and since then it has found various adherents, who have easily elaborated many arguments in favor of their proposition. Very recently Walton[8] and Krewer[9] have again argued the point. Walton modifies, on the basis of a careful analysis of reported cases, the usually accepted conception of the process, and defines it as a form of toxic peripheral neuritis. This conclusion he bases on the report of a case of his own and a study of other cases, with reference particularly to disturbances of sensation and electrical reaction. Worcester,[10] in a later publication, is inclined to agree with Walton. Krewer is absolutely confident of his position, that Landry's paralysis is nothing other than the second and third stages of a chronic multiple neuritis, which through continuity of structure has invaded the spinal cord. He further thinks that a new factor usually in the form of an infectious disease, must supervene on a pre-existing polyneuritis to induce the symptoms of Landry's paralysis. Four of the writer's cases are described in this paper; one of the patients lived and the other three fatal cases strongly suggest alcohol as an important etiological factor. Marked changes of a degenerative character were found both in the peripheral nerves and the cord, hence according to the author, Landry's paralysis is peripheral neuritis.[11] That patients die of rapidly progressive neuritis is unquestioned, but why call such cases Landry's paralysis, and then assume that we have discovered its pathological anatomy?[12] This is evidently a begging of the entire question.

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[8] Walton, Bost. Med. and Surg. Jour., Dec. 26, 1895. It should be stated that in a recent conversation with one of us, Dr. Walton said that he had changed his opinion regarding neuritis and Landry's paralysis. His present standpoint is not known to us.

[9] Krewer, Ztschft. f. klin. Med., 22, 1897, p. 115.

[10] Worcester, JOUR. NERV. AND MENT. DIS., May, 1898.

We quote these facts merely to illustrate our point that Landry's paralysis as a circumscribed group of symptoms has come to grief. The tendency has become quite irresistible to group under this convenient heading various clinical phenomena, between which no adequate bond of union has yet been found. Under these circumstances it might be expected that post-mortem study would throw light on the matter, and supply the desired facts which should make of Landry's paralysis a separate disease process.

This hope is by no means justified by a study of the more recent literature of the pathological changes. So far as we are able to discover, there is simply nothing characteristic about any of the lesions described by various writers. The more we look the more we find, undoubtedly, but no one lesion or series of lesions is explanatory. Mills and Spiller,[13] for example, after an exceedingly critical analysis of the whole question, and on the basis of a carefully studied case, reach the following general conclusions: that a type of disease, as described by Landry, exists; that transitional forms occur, making the diagnosis between Landry's paralysis, polyneuritis and myelitis difficult; that it is possible in some cases that no lesions exist; that Landry's paralysis may be due to myelitis alone; that polyneuritis may be present, usually accompanied by changes in ventral horn cells; that in some cases the entire peripheral motor neurone may be attacked at the same time by the poison of the disease. It appears from these statements that Mills and Spiller are inclined to be liberal in the interpretation of the pathological anatomy of what they still call Landry's paralysis. Inasmuch as Landry's paralysis may be due to each of these lesions, it is apparent that it is not due exclusively to any one lesion, and hence lacks coherence from the point of view of its pathological anatomy.

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[11] We confess to a certain scepticism of Krewer's pathological findings in general from the following statement, p. 123: "Im Endoneurium finden wir eine Hyperplasie der Neurogliazellen, ohne jedoch dieselbe als eine Zelleninfiltration ansehen zu können."

[12] A patient, for example of alcoholic tendencies under the observation of one of us at the Long Island Hospital, Boston, was taken with a gradually progressive muscular weakness, especially marked in the legs, associated with certain sensory disorders. The condition was progressive; the patient became almost completely paralytic and death resulted two months after the onset of symptoms. Were one so disposed, it might be possible to lay stress on the progressive motor weakness ending in death, and call the disease Landry's paralysis, instead of a general affection of the nervous system resulting from alcohol. We suspect that Krewer's cases were of this type.

[16] Diller and Meyer, *loc. cit.*

Bailey and Ewing[14] conclude from their work that the commonest seat of the lesion is within the cord or oblongata, though it may be present in the cortex or in the nerve roots. They go on to say: "When in the spinal cord the lesion is that of acute anterior poliomyelitis—namely, an acute exudative inflammation, following the distribution of the central branch of the anterior cerebral artery, with cellular infiltration of the circumvascular sheaths, degeneration of ganglion cells, loss of structural elements, and with or without degeneration of the anterior roots." These writers are doubtful of the peripheral lesion, and admit that the disease may run a fatal course without demonstrable histological changes in the nervous system. This is a perfectly candid statement that Landry's paralysis is poliomyelitis in the majority of cases, which widens our pathological anatomy, but unfortunately renders still more vague our terminology.[15]

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[14] Bailey and Ewing, *loc. cit.*

[15] The following case, to be reported in detail in a subsequent paper, is of interest in this connection: A man of 25, previously well, after several days of vague discomfort, lost the use of his legs, and subsequently of his arms to a certain degree; sensation was only very slightly disturbed; the paralysis was flaccid. After three months, during which he improved slightly, he died. Autopsy showed a marked congestion of both ventral horns throughout the whole length of the cord, which microscopic examination proved to be due to a destructive inflammation. Nissl sections showed many damaged and some intact

Krewer, already quoted, is confident that Landry's paralysis is a subacute chronic polyneuritis (whatever that may be), and an acute diffuse degenerative myelitis. He, of course, substantiates his view by autopsy and microscopic examination. This is quite opposed to Bailey and Ewing, and, in fact, to most other observers, for however often peripheral degenerations have been found, a true neuritis has been exceedingly rare.

Meyer[16] makes the following pertinent remarks: "Landry's paralysis is a symptom-complex without quite uniform post-mortem findings. There may be cases of very rapid course in which the naked eye, and even microscopical examinations with the help of older methods, does not reveal any lesion. With the improvement of the technique, these cases have become very rare, and there is reason enough to believe that the most recent technical proceedings would have demonstrated changes in most cases. Hereby one of the most important items that induced Landry to describe this symptom-group as a disease, *sui generis*, has fallen; the post-mortem findings can no longer be said to be negative, but they are at least not quite uniform." This is a very tentative statement of what is becoming more and more apparent as investigation progresses. We would simply go a step further and say that the lack of uniformity in the post-mortem findings is much more conspicuous than the similarities. Meyer, for example, in this same paper, calls attention to a degeneration of the pyramidal tracts as one of the lesions, a fact, in itself, of great interest, but quite out of accord with our preconceived ideas of the pathological anatomy of the disorder. On the other hand, in spite of perfected technique, lesions are, in certain cases, still undiscoverable. A case of this sort is described by Girandeau and Lévi,[17] who, in spite of a complete examina-

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cells. The diagnosis during life had been Landry's paralysis. According to Bailey and Ewing it was, but to our minds a better service would be rendered by calling it what the lesion demonstrated it to be, an acute poliomyelitis. Certainly a pathological term is to be preferred to a purely descriptive one, and that of highly inexact meaning. (Compare this case with one of those reported by J. J. Thomas, *loc. cit.*)

[16] Mills and Spiller, *loc. cit.*

[17] Girandeau and Lévi, "Un cas de paralysie ascendante aigue sans lesion histologique des nerfs et de la moelle." Rev. Neurologique, Oct. 15, 1898, p. 669.

tion, using the Nissl method, found nothing abnormal. Thomas[18] is inclined to the view that the essential feature of the process is an affection of the motor (spinal) neurone of the first order, which, however, may vary within very wide limits, depending upon the intensity of the infection. On the one hand these cases are very similar to acute anterior poliomyelitis, and on the other show changes of the slightest character in the motor elements. In general, Thomas thinks that the evidence "seems to justify us in retaining this collection of symptoms as forming a separate disease, clinically at least." This is a conservative statement of the case, and is merely open to the criticism that knowledge, on the whole, is not advanced by postulating groups of symptoms as diseases, particularly when it appears possible to discover an anatomical basis for such symptoms. In this matter of Landry's paralysis we are bound to find some essential connection between lesion and symptom, or else renounce the disease even as a clinical entity. This attitude the rapidly accumulating investigations on the subject are forcing upon us. We are still in the dark as to any fundamental and necessary association between the pathological findings and the observed symptoms.

It would be easy to multiply examples from the literature of discrepancies in the post-mortem appearances of the nervous system, in persons dying of what we have been in the habit of calling Landry's paralysis. The instances already given, however, seem quite sufficient to demonstrate the fact that there is absolutely no unity in these findings, and that, therefore, a causal relationship between them and the generally accepted symptomatology cannot be maintained.

In the absence of evidence tending to establish the individuality of the affection, both on the clinical and pathological side, it may be thought possible that a study of etiology will throw light on the matter, and supply the necessary link between cause and effect. This is, however, most certainly not the case. The vaguest part of the whole picture lies in the discovery of a constant etiological factor. To say that the disorder is due to an infection of some sort, or follows infectious diseases, does nothing to differentiate it from all the other recognized infectious processes, which give rise to totally different symptoms. Nor is our knowledge advanced by saying that it is a toxemia, unless we can also say what sort of a toxemia it is. These sweeping statements do little more than restate the problem in somewhat more exact language, but are not in the least explanatory of the peculiar symptoms which are claimed to be characteristic of the disease. The search for pathogenic organisms has also failed to show any relationship whatever between the bacteria found and the histological changes. Organisms described, for example, by Baumgarten, Centanni, Eisenlohr, and Piccinino, as occurring in the nervous system of patients dying of Landry's paralysis, are interesting as facts, but have as yet absolutely no etiological significance, for the reasons that they are not commonly found, that they vary widely in characteristics when found, and that they cannot be brought into causal relationship with the lesions or symptoms, by the recognized bacteriological experiments. As a matter of fact, beyond the mere statement that Landry's paralysis is probably due to a somewhat selective toxemia, our knowledge of the etiology does not go. The identity of the disease is rather dissipated than established by such a statement, inasmuch as it is thereby simply brought into the category of a great number of processes, which are awaiting completer investigation before they can be accorded the dignity of definite diseases, in the sense that diphtheria, for example, is a definite disease.

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[18] Thomas, *loc. cit.*, pp. 158-161.

So-called Landry's paralysis is a particularly good example of a large class of disorders of function, having a certain superficial uniformity of manifestation in which observers have continually inclined to lay undue stress on similarities, and to ignore dissimilarities, in the absence of any fundamental criterion of comparison. On the basis of an acute clinical, and of necessity highly superficial, pathological observation made many years ago by Landry, subsequent observers, led largely by a name, have been attempting to force into the narrow clinical conception outlined by Landry various symptoms, none of them fundamental and supported by no constant pathological findings, and still less by a definite etiology. This attempt, to our minds, has signally failed at every point, as we have attempted to show; the so-called disease has, with each new investigation, lost something of its coherence, when looked at from a critical point of view, and awaits a new classification under a broader conception of disease, for which our advancing knowledge is gradually preparing us. Evidently this new classification must be on the basis of etiology, and this applies not only to Landry's paralysis, but also to many other affections of the nervous system. One matter that must particularly impress itself upon the student of the pathological anatomy of the nervous system is the impossibility of sharply defining the limitations of anatomical changes in various text-book diseases. It is safe to say that such changes are always broader than the symptoms, which they are supposed to explain, as shown, for example, in the two well-marked diseases on the clinical side of tabes and paralytic dementia. We are too apt to be satisfied when we find alterations of structure, which we think explain our superficially observed symptoms, and too little inclined to search for symptoms of which pathological findings should lead us to expect the existence. A still commoner error, and one largely due to our defective knowledge, is our neglect of etiology. To define the cause of diseased tissues and abnormal symptoms is the ultimate work of the scientific medical investigator, and until that is done our classifications must not be dogmatic. Tuberculosis and many of the other germ diseases have yielded to this demand, with what result for exact medicine we know. For obvious reasons our knowledge of cause in the broad sense has lagged behind in relation to the nervous system, and we find ourselves postulating all sorts of diseases from symptoms alone, quite regardless of the broader conceptions which it should be the function of our scientific training to develop. However necessary this may be as a starting point of investigation, it is desirable to remember that conclusions based on such observation should always be tentative and not final. Landry's paralysis is a flagrant example of the failure to recognize this fact. We have persistently clung to a name and demanded that the symptoms which it represented should conform to our preconceived idea, rather than be merged in a larger idea of the fundamental process. If this were done and the term "Landry's paralysis" dropped as indicative of a special form of disease, no doubt our progress toward a reasonably scientific classification would be materially advanced. Bright's disease has gradually given place to "nephritis," a bad term still, but a great improvement; why should not "Landry's paralysis" give place to an inclusive term of definite significance, which shall be broad enough to embrace the whole contradictory array of symptoms and pathological findings?

From the foregoing discussion we feel justified in concluding: that inasmuch as there is no essential constancy in the clinical symptoms, nor in the pathological findings of so-called Landry's paralysis, and inasmuch as the etiology is wholly vague, it is probable that the affection does not represent in itself a process to which the term "disease" may properly be applied, and that, therefore, it is desirable to drop the term, as unnecessary and misleading.[19]

LE TRAITÉMENT DE QUELQUES TROUBLES TROPHIQUES DU PIED ET DE LA JAMBE PAR LA DÉNUDATION DE L'ARTÈRE FEMORALE ET LA DISTENSION DES NERFS VASCULAIRES (Treatment of Trophic Lesions of the Foot and Leg by Exposure of the Femoral Artery and Evulsion of the Vasa Vasorum). Jaboulay (Lyon Médical, 31, 1899, p. 467).

The above named surgeon is nothing if not original—not to say bizarre—in his contributions to surgery of the nervous system. In the case of an alcoholic and syphilitic man of forty-five years, who presented several small sloughing spots on the right foot and a cyanotic area on the right leg, all of three weeks' duration, and who suffered from pain in the second toe and in the leg, he exposed the femoral artery in Scarpa's triangle and tore away two nerves which accompanied the artery for about 15 cm. The following day a blister 5 cm. in diameter appeared on the sole of the foot, involved the entire arch, and broke on the tenth day revealing an ulcerated surface which was five weeks in healing. In the meantime, the original small sloughs separated and the ulcers healed. At the ends of six weeks the patient was discharged, the sores healed, but with pain in the second toe. The author's explanation of the result is quite as fantastic and immature as was the operation.
PATRICK.

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[19] Since this manuscript left our hands, a paper on Landry's Paralysis has appeared by Knapp and Thomas, JOUR. OF NERV. AND MENT. DIS., February, 1900, p. 74. It is, in general, a reiteration of the opinions before expressed by one of the authors (Thomas), and in our opinion in no way controverts the position we have taken in this paper.

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